areas. The South Coast Area has been designated as extreme [2]; therefore, this area was subject to the RACT fix-up requirement and the May 15, 1991 deadline.

The State of California submitted many revised RACT rules for incorporation into its SIP on February 24, 1995, including the rule being acted on in this document. This document addresses EPA's proposed action for SCAQMD Rule 1164. SCAQMD adopted Rule 1164 on January 13, 1995. This submitted rule was found to be complete on March 10, 1995 pursuant to EPA's completeness criteria that are set forth in 40 CFR part 51 Appendix V [3] and are being proposed for approval into the SIP.

SCAQMD Rule 1164 controls the VOC emissions during the operations of semiconductor manufacturing. VOCs contribute to the production of ground-level ozone and smog. This rule was adopted as part of the SCAQMD's efforts to achieve the National Ambient Air Quality Standard (NAAQS) for ozone and in response to EPA's SIP-Call and the section 182(a)(2)(A) CAA requirement. The following is EPA's evaluation and proposed action for this rule.

EPA Evaluation and Proposed Action

In determining the approvability of a VOC rule, EPA must evaluate the rule for consistency with the requirements of the CAA and EPA regulations, as found in section 110 and part D of the CAA and 40 CFR part 51 (Requirements for Preparation, Adoption, and Submittal of Implementation Plans). The EPA interpretation of these requirements, which forms the basis for today's action, appears in the various EPA policy guidance documents listed in footnote 1. Among those provisions is the requirement that a VOC rule must, at a minimum, provide for the implementation of RACT for stationary sources of VOC emissions. This requirement was carried forth from the pre-amended Act.

For the purpose of assisting state and local agencies in developing RACT rules, EPA prepared a series of Control Technique Guideline (CTG) documents. The CTGs are based on the underlying requirements of the Act and specify the presumptive norms for what is RACT for specific source categories. Under the CAA, Congress ratified EPA's use of these documents, as well as other Agency policy, for requiring States to ''fix-up'' their RACT rules. See section 182(a)(2)(A). EPA has not yet developed a CTG to outline control requirements for the semiconductor manufacturing source category. Therefore, interpretations of EPA policy are found in the Blue Book, referred to in footnote 1, and the Region IX/CARB document entitled, *Guidance Document for Correcting VOC Rule Deficiencies*. In general, these guidance documents have been set forth to ensure that VOC rules are fully enforceable and strengthen or maintain the SIP.

SCAQMD Rule 1164—Semiconductor Manufacturing includes the following significant changes from the current SIP:

- Section (b)(1) includes an appropriate definition for *approved emission control system* which requires the system to have an overall efficiency of at least 90 percent.
- Section (b)(14) includes an equation to determine VOC composite partial pressure.
- Other definitions were added or altered for clarity.
- Sections (e)(1) and (e)(2) list the test methods for determining VOC content of any VOC-containing materials or vapors. These methods include EPA Test Method 24, SCAQMD Method 303, SCAQMD Method 304, SCAQMD Method 308.
- Section (e)(3) includes test methods for determining the efficiency of the emission control systems. These methods include the EPA method cited in 55 **Federal Register** 26865, EPA Test Methods 25, 25A, 18, ARB 422, or SCAQMD Method 25.1.
- Section (e)(4) ensures that a violation of any requirement of this rule established by any one of the specified test methods shall constitute a violation of the rule when more than one test method is specified for any testing.

EPA has evaluated the submitted rule and has determined that it is consistent with the CAA, EPA regulations, and EPA policy. Therefore, SCAQMD Rule 1164 is being proposed for approval under section 110(k)(3) of the CAA as meeting the requirements of section 110(a) and Part D.

Nothing in this action should be construed as permitting or allowing or establishing a precedent for any future request for revision to any state implementation plan. Each request for revision to the state implementation plan shall be considered separately in light of specific technical, economic, and environmental factors and in relation to relevant statutory and regulatory requirements.

Regulatory Process

Under the Regulatory Flexibility Act, 5 U.S.C. 600 et seq., EPA must prepare a regulatory flexibility analysis assessing the impact of any proposed or final rule on small entities. 5 U.S.C. 603 and 604. Alternatively, EPA may certify that the rule will not have a significant impact on a substantial number of small entities. Small entities include small businesses, small not-for-profit enterprises and government entities with jurisdiction over populations of less than 50,000.

SIP approvals under sections 110 and 301 and subchapter I, part D of the CAA do not create any new requirements, but simply approve requirements that the State is already imposing. Therefore, because the Federal SIP-approval does not impose any new requirements, it does not have a significant impact on any small entities affected. Moreover, due to the nature of the Federal-state relationship under the CAA, preparation of a regulatory flexibility analysis would constitute Federal inquiry into the economic reasonableness of state action. The CAA forbids EPA to base its actions concerning SIPs on such grounds. *Union Electric Co.* v. *U.S. E.P.A.*, 427 U.S. 246, 256–66 (S.Ct. 1976); 42 U.S.C. 7410(a)(2).

The OMB has exempted this action from review under Executive Order 12866.

List of Subjects in 40 CFR Part 52

Environmental protection, Air pollution control, Hydrocarbons, Intergovernmental relations, Ozone, Reporting and recordkeeping requirements, Volatile organic compound.

Authority: 42 U.S.C. 7401–7671q.

Dated: April 11, 1995.

Felicia Marcus,

Regional Administrator.

[FR Doc. 95–9709 Filed 4–19–95; 8:45 am]

BILLING CODE 6560–50–W

[2] The South Coast Area retained its designation of nonattainment and classified by operation of law pursuant to sections 107(d) and 181(a) upon the date of enactment of the CAA. See 55 FR 56694 (November 6, 1991).

[3] EPA adopted the completeness criteria on February 16, 1990 (55 FR 5830) and, pursuant to section 110(k)(1)(A) of the CAA, revised the criteria on August 26, 1991 (56 FR 42216).

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40 CFR Part 372

[OPPTS–400092; FRL–4946–2]

Monosodium Methanearsonate and Disodium Methanearsonate; Toxic Chemical Release Reporting; Community Right-to-Know

AGENCY: Environmental Protection Agency (EPA).

ACTION: Denial of petition.

SUMMARY: EPA is denying a petition to delist monosodium methanearsonate (MSMA, CAS No. 2163–80–6) and disodium methanearsonate (DSMA, CAS No. 144–21–8) from the reporting requirements under section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA). This action is based on EPA's conclusion that neither monosodium methanearsonate or disodium methanearsonate meet the deletion criteria of EPCRA section 313(d)(3). Specifically, EPA is denying this petition because: (1) Monosodium methanearsonate and disodium methanearsonate are known to cause toxic effects in experimental animals as a result of chronic exposure to either of these substances; and (2) monosodium methanearsonate and disodium methanearsonate can reasonably be anticipated to cause cancer in humans.

FOR FURTHER INFORMATION CONTACT: Maria J. Doa, Petitions Coordinator, 202–260–9592, for specific information regarding this document. For further information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Information Hotline, Environmental Protection Agency, Mail Stop 5101, 401 M St., SW., Washington, DC 20460, Toll free: 800–535–0202, Toll free TDD: 800–553–7672.

SUPPLEMENTARY INFORMATION:

I. Introduction

A. Statutory Authority

This action is issued under sections 313(d) and (e)(1) of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA), 42 U.S.C. 11023. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act (SARA) of 1986 (Pub. L. 99–499).

B. Background

Section 313 of EPCRA requires certain facilities manufacturing, processing, or otherwise using listed toxic chemicals to report their environmental releases of such chemicals annually. Beginning with the 1991 reporting year, such facilities also must report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the Pollution Prevention Act of 1990 (PPA), 42 U.S.C. 13106. Section 313 established an initial list of toxic chemicals that was comprised of more than 300 chemicals and 20 chemical categories. Section 313(d) authorizes EPA to add or delete chemicals from the list, and sets forth criteria for these actions. EPA has added and deleted chemicals from the original statutory list. Under section 313(e), any person may petition EPA to add chemicals to or delete chemicals from the list. EPA must respond to petitions within 180 days, either by initiating a rulemaking or by publishing an explanation of why the petition is denied.

EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479), to provide guidance regarding the recommended content and format for submitting petitions. On May 23, 1991 (56 FR 23703), EPA issued guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compound categories. EPA has also published a statement clarifying its interpretation of the section 313(d)(2) criteria for adding and deleting chemical substances from the section 313 list (59 FR 61439, November 30, 1994).

II. Description of Petition and Relevant Regulations

On October 18, 1994, EPA received a petition from the ISK Biosciences Corporation to remove monosodium methanearsonate (MSMA) and disodium methanearsonate (DSMA) from the list of toxic chemicals subject to the requirements of section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA). Specifically, the petition requests that MSMA and DSMA be excluded from the arsenic compounds category which is subject to annual release reporting requirements under EPCRA section 313. The petitioner contends that MSMA and DSMA should be deleted from the EPCRA section 313 arsenic compounds category because, in their opinion, the available data show that neither of these substances meet the criteria for inclusion on the list of EPCRA section 313 chemicals. The petitioner did not provide EPA with any of the studies cited in the petition.

MSMA and DSMA are organic arsenicals. EPA regulates arsenic and certain arsenic compounds under the Clean Air Act (CAA), Clean Water Act (CWA), Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), Resource Conservation and Recovery Act (RCRA), Safe Drinking Water Act (SDWA), and EPCRA. Arsenic emissions from smelters and other facilities are regulated under the CAA. Under the CWA, guidelines have been established controlling the environmental release of arsenic compounds for certain industrial categories. Reportable quantities have been established under CERCLA and CWA for arsenic and certain arsenic compounds. Under RCRA, EPA regulates arsenic as a hazardous constituent of waste. The SDWA limits arsenic in drinking water to a maximum level of 0.05 milligrams/liter (mg/L). EPA and the National Toxicology Program have classified inorganic arsenicals, including arsenate, as known human carcinogens.

III. EPA's Technical Review of Monosodium Methanearsonate (MSMA) and Disodium Methanearsonate (DSMA)

The technical review of the petition to delete MSMA and DSMA included an analysis of the chemistry, health, ecological and environmental fate data known for these substances and for methanearsonic acid (MAA), the un-ionized form of MSMA and DSMA. From a human health standpoint, MSMA and DSMA will exist largely as MAA (their un-ionized form) under acidic conditions, such as those found in the gastrointestinal tract. Also, following absorption into the systemic circulation, MSMA, DSMA, and MAA will exist in an identical ionized form at the physiological pH of 7.4, regardless of their route of administration. EPA and the ISK Biosciences Corporation (as indicated in their petition) believe, therefore, that mammalian toxicity data on MAA should be suitable to assess the toxicity of MSMA and DSMA in cases where such data on the latter two substances are not available.

A. Chemistry

Monosodium methanearsonate (CH_4AsO_3.Na; CAS No. 2163–80–6), also known as MSMA, and disodium methanearsonate (CH_3AsO_3.2Na; CAS No. 144–21–8), also known as DSMA, are the monosodium and disodium salts, respectively, of methanearsonic acid (also known as MAA). MSMA, DSMA, and MAA are often refered to as organic arsenicals, because they each contain a methyl ($–CH_3$) group. Both MSMA and DSMA are highly water soluble crystalline solids, and are used as herbicides for the postemergent control of grassy weeds in cotton, sugarcane, nonbearing orchards, citrus groves, lawns, turf, and in noncrop areas. The predominant use of MSMA and DSMA is for postemergent control of Johnsongrass and other grassy weeds prior to planting cotton.

B. Toxicological Evaluation

Information on the health and environmental effects of MSMA, DSMA, and MAA were obtained from the following sources: a 1993 Agency for Toxic Substances and Disease Registry

document entitled *Toxicological Profile for Arsenic (Update)* (Refs. 2, 15, and 30); a 1984 EPA document entitled *Health Assessment Document for Arsenic* (Ref. 7); a 1994 National Toxicology Program document entitled *Seventh Annual Report on Carcinogens: 1994 Summary* (Ref. 32); studies obtained from EPA's Office of Pesticide Programs (Ref. 8, 10, 12–14, 16 and 19–24); and studies found in the literature (Refs. 1, 3–6, 9, 11, 17, 18, 25, 26, 28, 29, and 31). Specifically, toxicological and related data on MSMA, DSMA, and MAA (the un-ionized or free acid form of MSMA and DSMA) were reviewed for evidence indicating: (1) Bioavailability and metabolism to inorganic arsenic; (2) acute toxicity; (3) chronic toxicity; (4) carcinogenicity; and (5) ecotoxicity.

1. *Bioavailability and metabolism*. Shah and co-workers investigated the absorption of MSMA and DSMA from the skin of young and adult rats (Ref. 1). Both substances were very poorly absorbed through the skin of all animals tested, particularly in the younger animals. No human studies pertaining to the dermal absorption of MSMA and DSMA were found. However, human and animal studies involving dermal exposure to organic arsenicals closely related to MSMA and DSMA indicate that these substances are poorly absorbed from the skin (Ref. 2).

Shariatpanahi and Anderson found that MSMA is readily absorbed from the gastrointestinal tract following oral administration of the substance to sheep and goats (Ref. 3). These investigators observed that 90 percent of the arsenic content of orally administered MSMA was excreted in the urine of test animals within 120 hours of administration. Small amounts were excreted in the feces. Arsenic accumulation in the tissues was low. It is noteworthy to point out that metabolism of MSMA to other forms of arsenic (e.g., inorganic) was not studied in this investigation, and only total arsenic concentrations were determined. Specific assays for MSMA or other specific arsenicals were not used. The results of this study were consistent with the results of another study, which investigated the absorption, distribution and elimination of MSMA in New Zealand white rabbits following multiple oral doses of the substance (Ref. 4).

A 1991 EPA study investigated the absorption, distribution, and elimination of radiolabeled MSMA ([^{14}C-methyl]MSMA) in rats (Ref. 8). Four groups of rats were used in this study. Each group consisted of male and female animals. One group received a single oral dose of [^{14}C-methyl]MSMA at 5 milligrams per kilogram (mg/kg), while another group received a single oral dose of 200 mg/kg. A third group received a single oral dose of MSMA at 5 mg/kg every day for 14 consecutive days, followed by a single oral dose of [^{14}C-methyl]MSMA. A fourth group received a single oral dose of MSMA at 5 mg/kg every day for 14 consecutive days, followed by a single intravenous dose of [^{14}C-methyl]MSMA at 5 mg/kg or a single oral dose of [^{14}C-methyl]MSMA at 5 mg/kg. In each of the test groups, the majority (79.7 to 97.4 percent) of administered [^{14}C-methyl]MSMA was excreted unchanged in the urine and feces within 7 days following dosing. Radiolabeled carbon dioxide ($^{14}CO_2$) was detected in all treated groups, and accounted for less than 0.5 percent of administered [^{14}C-methyl]MSMA. An unidentified metabolite, which accounted for 1.8 to 6.7 percent of administered [^{14}C-methyl]MSMA, was detected in the urine and feces of all test groups except the group receiving 200 mg/kg [^{14}C-methyl]MSMA orally. Another unidentified metabolite, accounting for 0.7 percent of administered [^{14}C-methyl]MSMA was found in only one of the test groups.

Buchet, et al., investigated the oral absorption and metabolism of MSMA in humans (Ref. 9). In this study four adult males were administered MSMA in a single oral dose equivalent to 500 micrograms of arsenic. The MSMA was well absorbed, and nearly 70 percent of the dose was excreted unchanged in the urine within 24 hours, while a small percentage was excreted in the urine as cacodylic acid (dimethylarsonic acid). Within 96 hours, 78.3 percent of the MSMA dose was excreted in the urine unchanged and approximately 13 percent was excreted in the urine as cacodylic acid. No inorganic arsenic metabolites were identified (Ref. 9).

Stevens and co-workers investigated the toxicity of DSMA in rats and mice exposed to the substance at aerosolized doses of 6.1 mg/L (for the rats) and 6.9 mg/L (for the mice) for 2 hours (Ref. 5). Total arsenic levels from body fluids or tissues were not determined, but the authors believed that some absorption of DSMA occurred from the lung.

2. *Acute toxicity*. Several rat oral median lethal dose (LD_{50}) values for MSMA and DSMA were found in the literature. For DSMA, the rat oral LD_{50} values, in mg/kg, are (male, female): 2,005, 1,842 (Ref. 10); and 928, 821 (Ref. 11). For MSMA, the rat oral LD_{50} values are 1,105 and 1,059 mg/kg for males and females respectively (Ref. 11). These data are consistent with rat median lethal dose data provided by the petitioner.

Neither DSMA or MSMA produced significant toxicity in rabbits when applied dermally at a dose of 2,000 mg/kg for 24 hours (Refs. 12 and 13). In the MSMA- treated group, however, there was evidence of decreased muscle tone noted in approximately 50 percent of the animals on observation days 5 through 9 (Ref. 13). By observation day 10, muscle tone was normal in all treated animals.

In a study investigating the acute inhalation toxicology of DSMA, mice and rats were placed in chambers and were exposed for 2-hours to experimental atmospheres containing DSMA in concentrations of at least 8.6 mg/L (Ref. 5). The animals were observed to have respiratory distress during the 2–hour exposure period, but recovered rapidly after removal from exposure. Respiratory irritation was the main toxicological effect observed. No mortality occurred in either species. These results are consistent with those of a similar DSMA inhalation study (Ref. 14). In the latter study, rats were exposed to experimental atmospheres of 6.0 mg/L DSMA for 4 hours. No deaths were noted during the 14-day post-exposure observation period. Clinical signs noted on the first day post-exposure included body weight loss and respiratory irritation. Lung discoloration in 40 percent of the animals was also noted (Ref. 14).

3. *Chronic toxicity*. Numerous studies investigating the chronic toxicity of inorganic arsenicals have been conducted. Relatively few studies, however, have investigated the potential for chronic toxicity of organic arsenicals such as MSMA, DSMA, and MAA. The limited amount of published mammalian toxicity data on these substances have been summarized (Ref. 15). In addition, the petitioner summarized unpublished chronic toxicity data that are available from EPA's Office of Pesticide Programs. Some of these studies will be briefly discussed here.

In a study investigating the health effects resulting from chronic administration of MAA, four groups of rats (each group consisting of 60 males and 60 females) were fed diets containing 0 (the control group), 50, 400, and 1,300 parts per million (ppm) of MAA for 104 weeks (Ref. 16). Mortality was significantly increased in animals fed diets containing 1,300 ppm MAA. Because of this increased mortality, the 1,300 ppm concentration was reduced to 1,000 ppm during week 53, and to 800 ppm at week 60. Animals in this group had acute gastrointestinal inflammation, ulceration and perforation of the large intestines, and

evidence of acute or chronic peritonitis. These observations were less evident in animals receiving diets containing 400 ppm MAA. A reduction in the weight of the thyroid glands was noted in female rats receiving the 1,300 ppm and 400 ppm MAA diets, and in male rats receiving 400 ppm MAA. Thickening of the thyroid follicular epithelium was noted in both sexes receiving the 1,300 and 400 ppm MAA diets. An increased incidence of parathyroid adenomas may have occurred in male rats receiving the 1,300 and 400 ppm MAA diets. This observation is discussed in greater detail in unit III.B.4 below.

Jaghabir and co-workers investigated the health effects of low dose MSMA exposure in white rabbits (Ref. 17). Three groups of rabbits were used in this study. The first group consisted of four rabbits, which were administered MSMA orally once a day for 40 days at a dose of 5 mg/kg. The second group consisted of two animals, which were administered MSMA at a dose of 10 mg/kg orally for 40 days. The third group (also consisting of two animals) was similarly administered MSMA at a dose of 20 mg/kg. A control group of two animals was also used. All animals were euthanized and examined at the end of the 40-day test period. Post-mortem examination revealed distension and hyperemia of the digestive tract, intestinal wall fragility, enlargement of the kidneys, and intense peripheral hyperemia of the livers of all animals administered MSMA. Histopathological findings revealed hepatic cellular degeneration, periportal inflammation, renal tubular nephrosis, interstitial nephritis and vascular hyperemia. These observations are consistent with the observations of similar investigations cited in the study (Ref. 17), and indicate that low dose exposure to MSMA can result in tissue damage.

Results from several studies suggest that MSMA and DSMA may cause developmental and reproductive toxicity. In an investigation reported by Prukop and Savage (Ref. 18) it was observed that mice administered MSMA at doses of either 11.9 or 119 mg/kg orally three times a week for 10 weeks had decreased reproductive capabilities (males) and altered reproductive behavior (females). In another study, groups of beagle dogs were administered MAA at 0 (control), 2.5, 8 or 40 mg/kg/day for 1 week, followed by administration of 0 (control animals), 2, 8, or 35 mg/kg/day for an additional 51 weeks (Ref. 19). Decreased body weight gain occurred in male dogs that received the 35 mg/kg/day dose, and in females that received the 8 or 35 mg/kg/day doses. The incidence of female animals showing no corpora lutea were increased in the 35 mg/kg/day animal test group when compared to control animals (Ref. 19).

In another study, groups of inseminated New Zealand white rabbits were administered MAA orally at doses of 0 (control animals), 1, 3, 7, and 12 mg/kg/day during days 7 thru 19 of gestation (Ref. 20). Maternal toxicity at 12 mg/kg/day was characterized by abortion and decreases in mean absolute body weight, body weight gain, and food consumption. Decreases in body weight gain and food consumption were also noted in the 7 mg/kg/day test group. An increased incidence of skeletal variations was noted in the offspring of animals administered MAA at 12 mg/kg/day. These skeletal variations consisited of increased numbers of ribs and thoracic and lumbar vertebrae (Ref. 20).

In a multigeneration toxicity study, groups of male rats were fed MAA at doses of 0 (control group), 5.8, 17.8, or 63.5 mg/kg/day, and groups of female rats were fed 0 (control group), 7.5, 22.5, and 77.6 mg/kg/day for 14 weeks. Animals were mated, and mated females continued to receive MAA throughout gestation and lactation periods. Among other toxic effects noted in the 63.5 (males) and 77.6 (females) mg/kg/day dose groups, decreased pregnancy rates, male fertility rates, and decreased weights of the prostate and testes also occurred for parenteral generations F0 and F1 (Ref. 21).

A study was conducted in which MSMA was administered orally to pregnant female rats at doses of 0, 10, 100, or 500 mg/kg once daily on gestation days 6 through 15. No developmental effects were noted in the offspring of animals receiving 10 or 100 mg/kg MSMA. Decreased body weight gain and food consumption were noted in animals receiving 500 mg/kg MSMA. The fetuses of this test group had lower mean fetal body weights when compared to control animals (Ref. 22).

Based on the results of the animal studies discussed in the preceding paragraphs, EPA has determined that chronic exposure to either MSMA or DSMA can reasonably be anticipated to cause gastrointestinal toxicity, thyrotoxicity, nephrotoxicity, hepatotoxicity, and developmental and reproductive toxicity in humans.

4. *Carcinogenicity*. Data regarding the carcinogenic potential of MSMA, DSMA, or MAA are extremely limited. In a study involving chronic administration of MAA, four groups of rats, each group containing 60 males and 60 females, were fed diets containing 0 (the control group), 50, 400, and 1,300 ppm of MAA for 104 weeks (Ref. 16). Because of excessive mortality, the 1,300 ppm concentration was reduced to 1,000 ppm during week 53, and to 800 ppm at week 60. An increased incidence of parathyroid adenomas was observed in males receiving the 1,300 ppm (4/45) and 400 ppm (4/53) MAA diets, and in females (4/45) receiving the 1,300 ppm MAA diets. Evidence of parathyroid adenoma was also found in 1 of 52 male control rats. The increased incidence of parathyroid adenomas in the treated groups was found to be statistically significant relative to the control animals.

As stated previously, cacodylic acid (dimethylarsonic acid, CAS No. 75–60–5) is a known human metabolite of MSMA: Buchet and co-workers found that in human volunteers approximately 13 percent of an orally-administered dose of MSMA is converted into cacodylic acid (Ref 9). EPA has recently categorized cacodylic acid as a Group B2 or probable human carcinogen (Ref. 23). EPA's classification of cacodylic acid as a Group B2 carcinogen was based on the results of two studies. The first was a 2-year dietary feeding study in male and female rats receiving cacodylic acid at doses of 0, 2, 10, 40, and 100 ppm. An increase in urinary transitional cell bladder tumors with hyperplasia was noted in both sexes. The second study was a two year feeding study in which mice were fed diets containing 0, 8, 40, 200, and 500 ppm cacodylic acid. An increase in fibrosarcomas was noted in female mice fed 500 ppm cacodylic acid (23).

EPA is unaware of any human epidemiological studies pertaining to MSMA, DSMA or MAA and cancer. However, because MAA has been associated with a possible increased incidence of parathyroid adenomas in experimental animals, and cacodylic acid (a known human metabolite of MSMA) is categorized by EPA as a probable human (B2) carcinogen, EPA believes that it is reasonable to assume that MSMA, DSMA, and MAA may be potential human carcinogens.

5. *Ecotoxicity*. EPA has calculated a bobwhite quail oral LD_{50} of 425.2 mg MSMA/kg (Ref. 24). This value was based on 51 percent active ingredient (MSMA) in the test material. EPA concluded from this study that MSMA is moderately toxic to bobwhite quail. Based on the same study, the petitioner gave an LD_{50} value of MSMA in bobwhite quail as 834 mg/kg. This value, however, was not adjusted to take into account that the test product contains only 51 percent MSMA. Moffett, et al., have investigated the

toxicity of MSMA and DSMA in honeybees (Refs. 25 and 26). In one of the studies, MSMA was sprayed onto honeybees at a rate of 4 lb/acre in a carrier volume of 20 gallons/acre (Ref. 25). Mortalities were monitored for 14 days. Bee mortalities reached 50 percent after only approximately 2 days. Consequently, the investigators concluded that MSMA is highly toxic to honeybees (Ref. 25). In the other study MSMA and DSMA were fed to newly emerged honeybees in a 60 percent sucrose syrup (Ref. 26). Half-lives (i.e. the number of days for 50 percent mortality to occur) for MSMA and DSMA were 5.4 and 4.4 days at 100 parts per million by weight (ppmw) concentrations, and 2.5 and 1.2 days at 1,000 ppmw, respectively. The investigators concluded that both chemicals are ''extremely toxic'' at 100 and 1,000 ppmw. Of the 14 herbicides tested in this study, MSMA and DSMA were found to be the most toxic to honeybees (Ref. 26). EPA does not yet have toxicity criteria for honeybees in EPA's Draft Hazard Assessment Guidelines for Listing Chemicals on the Toxic Release Inventory (Ref. 27). EPA believes, however, that the results of the studies described above strongly indicate that MSMA and DSMA are quite toxic to honeybees.

The petitioner stated that for MSMA the acute median effective concentration (EC_{50}) producing lethality in the freshwater alga *Selenastrum capricornutum* is 7.6 mg/L. The petitioner concluded (page 68 of the petition) from this and other information that MSMA and DSMA are ''* * *.not particularly toxic to aquatic life * * *.'' However, based on the draft criteria developed by EPA to assess the hazard of chemical substances, EPA considers MSMA to be moderately toxic to aquatic life because the algal acute EC_{50} value for MSMA is between 100 micrograms per liter (ug/L) and 10 mg/L, the EC_{50} range considered by EPA to be moderately toxic for aquatic biota (Ref. 27). Other aquatic toxicity test data mentioned in the petition also indicate MSMA and DSMA are moderately acutely toxic (i.e., have EC_{50} or LC_{50} [median lethal concentration] values between 100 ug/L and 10 mg/L) to aquatic biota. The 96-h LC_{50} of MSMA in bluegill, for example, is 4.2 mg/L.

EPA obtained MSMA and DSMA aquatic toxicity data not mentioned by the petitioner (Ref. 28). The 28-day daphnid LC_0 (zero percent lethal concentration) value for DSMA is 0.83 mg/L. The LC_0 for DSMA in two species of invertebrates (a snail and a stonefly) and rainbow trout was found to be 0.97 mg/L (Ref. 28). A 28-day LC_{40} (40 percent lethal concentration) value of 0.97 mg/L DSMA was reported for a gammarid amphipod invertebrate. In bluegills, the 96-h LC_{50} for MSMA was found to be 1.9 mg/L. These data indicate that the toxicity of MSMA and DSMA to aquatic species is greater than that implied by the petitioner.

C. Environmental Fate

Anthropogenic input of arsenic into the environment occurs through smelting, coal burning, and the use of arsenical herbicides (e.g., MSMA and DSMA) (Refs. 29 and 30). Numerous investigators have studied the environmental fate of arsenic-containing substances, including MSMA and DSMA. Results from these studies have been summarized (Refs. 29, 30, and 31). Arsenic-containing substances such as MSMA, DSMA, and MAA undergo chemical and biochemical transformations in the environment that include oxidation, reduction, and methylation. These transformations are largely controlled by soil, sediment absorption/desorption processes, and affect the overall environmental distribution of arsenic-containing substances (Refs. 29, 30, and 31). Following their release into the environment, MAA, MSMA, and DSMA bind reversibly to ferrous and aluminum oxides contained on the surfaces of clay particles of soils and sediments. The bound form of these substances are insoluble in water, and exist in equilibrium with their unbound, soluble forms in the water present in soils and sediments. While unbound, MAA, MSMA, and DSMA undergo a cascade of biotic transformations that include oxidation, reduction, methylation, and demethylation (Ref. 31). Specifically, MAA, MSMA, and DSMA undergo oxidative demethylation to arsenate (H_2AsO_4-), an inorganic form of arsenic, and reductive methylation to cacodylic acid. The arsenate can be methylated back to MAA, and the two species will exist in equilibrium. Cacodylic acid can undergo further methylation to dimethylarsine or trimethylarsine, which will exist in equilibrium with cacodylic acid. These alkylarsine products volatilize from the soils and waters in which they were formed and enter the atmosphere. While in the atmosphere the alkylarsines can be transported to other locations, and the transformation cascade is repeated: the alkylarsines are oxidized back to cacodylic acid, MAA, and arsenate (Refs. 29–31). Thus, anthropogenic releases of MSMA or DSMA may indirectly lead to increased arsenic concentrations in areas where direct anthropogenic releases of these substances do not occur (Refs. 29–31). Terrestrial plants may accumulate arsenic-containing substances by root uptake from soils or by absorption of airborne arsenic deposited on plant leaves (Ref. 30).

The predominant form of arsenic in surface waters (e.g., drinking waters, sea waters, etc.) is usually arsenate (H_2AsO_4-), an inorganic form of arsenic. Arsenate in surface waters can result from (or enter into) the transformation cascade described in the preceding paragraph. Above average exposure of the general population to arsenic from drinking waters is possible in areas of high natural arsenic levels in ground waters, or elevated arsenic levels in drinking waters due to industrial discharges, application of arsenic-containing pesticides, or leaching from hazardous waste facilities (Ref. 30). Individuals living in the vicinity of large smelters and other industrial emitters of arsenic substances may be exposed to greater than average amounts of arsenate as a result of environmental transformation of organic (e.g., MSMA or DSMA) or inorganic arsenic substances to arsenate (Ref. 30).

Arsenate is an inorganic form of arsenic. An association between skin cancer and consumption of drinking water containing inorganic arsenic has been observed and confirmed (Ref. 32). Epidemiologic studies in areas where drinking waters containing inorganic arsenic concentrations ranging from 0.35 to 1.14 mg/L indicate elevated risks for cancers of the urinary bladder, kidney, skin, liver, lung, and colon in both men and women (Ref. 32). Increased incidences of cancer in individuals occupationally exposed to inorganic forms of arsenic have also been confirmed (Ref. 32). Because of these findings and the findings from other studies regarding human exposure to inorganic forms of arsenic and increased incidences of cancer, the National Toxicology Program categorizes arsenic and certain arsenic compounds (e.g., arsenate) as known human carcinogens (Ref. 32). EPA also categorizes inorganic arsenicals, including arsenate, as known human (Group A) carcinogens. The categorization by EPA of cacodylic acid as a Group B2 (probable human) carcinogen was discussed in unit III.B.4. above. Thus, releases of MSMA or DSMA into the environment will lead to the formation of arsenate and cacodylic acid, which have been categorized by the National Toxicology Program and EPA as carcinogens.

D. Technical Summary

MSMA and DSMA are highly water soluble organic arsenicals that are used as herbicides for the postemergent control of grassy weeds. MSMA and DSMA are poorly absorbed from the skin and lung, and well absorbed from the gastrointestinal tract. In the gastrointestinal tract, both MSMA and DSMA are expected to exist largely as MAA. Based on human and animal studies, MAA, MSMA, and DSMA are expected to be completely absorbed and widely distributed in humans following oral administration. In humans, MSMA is excreted largely unchanged in the urine, and approximately 13 percent is metabolized to cacodylic acid. MSMA and DSMA are not believed to be metabolized to inorganic arsenicals in humans.

The mammalian LD_{50} values of MSMA and DSMA following acute oral exposure are quite high, indicating that these substances have a low order of acute lethality. Some animal studies indicate, however, that chronic exposure to lower doses of MSMA or DSMA produce gastrointestinal toxicity, thyrotoxicity, nephrotoxicity, hepatotoxicity, developmental and reproductive toxicity. Data regarding the carcinogenic potential of MSMA, DSMA, or MAA are extremely limited. A suggestion of an increased incidence of parathyroid adenomas was observed in rats administered MAA in their diets. Cacodylic acid, a known human metabolite of MSMA, is categorized by EPA as a Group B2 (probable human) carcinogen. Because MSMA and, presumably, DSMA are converted into cacodylic acid, MSMA and DSMA may also be carcinogenic in humans.

MSMA and DSMA are moderately toxic to terrestrial and aquatic species that include, among others, bobwhite quail, honeybees, freshwater algae, fish, and daphnids.

In the environment, MSMA, DSMA, and MAA undergo a cascade of chemical and biochemical transformations that are controlled by soil, sediment adsorption/desorption processes. In this cascade, MSMA, DSMA, and MAA are converted into arsenate (inorganic arsenic), cacodylic acid, dimethylarsine and trimethylarsine. Inorganic arsenicals, including arsenate, are categorized by the National Toxicology Program and EPA as known human carcinogens. In addition, cacodylic acid is categorized by EPA as a Group B2 or probable human carcinogen.

IV. Rationale for Denial

EPA is denying the petition to delete MSMA and DSMA from the section 313 list of toxic chemicals. This denial is based on the Agency's determination that MSMA and DSMA: (1) May cause chronic toxic effects in humans; and (2) are potential carcinogens. In regard to the latter point, EPA has determined that because MSMA and, undoubtedly, DSMA are metabolized in humans to cacodylic acid (a probable human carcinogen), it is reasonable to assume that MSMA and DSMA are also probable human carcinogens. In addition, it has been demonstrated that MSMA and DSMA are converted into arsenate (an inorganic arsenic) and cacodylic acid in soils and sediments. Inorganic arsenics, including arsenate, are categorized by the National Toxicology Program and EPA as known human carcinogens. EPA concludes that MSMA and DSMA meet the EPCRA section 313(d)(2)(B) criteria because they can reasonably be anticipated to cause cancer in humans as a result of their metabolism to cacodylic acid or their environmental conversion to cacodylic acid and arsenate. Thus, in accordance with EPCRA section 313(d)(2), EPA has determined that MSMA and DSMA exhibit high chronic toxicity and, therefore, should not be deleted from the section 313 list of toxic chemicals.

EPA's denial of the petition to delist MSMA and DSMA from the section 313 list of toxic chemicals is based, in part, on the conversion of these substances to substances that are regarded as being either known or probable human carcinogens, and is consistent with past Agency decisions regarding section 313 delisting petitions. [See, e.g., Chromium (III) Oxide (56 FR 58859, November 22, 1991)]

V. References

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VI. Administrative Record

The record supporting this decision is contained in docket control number OPPTS–400092. All documents, including an index of the docket, are available to the public in the TSCA NonConfidential Information Center (NCIC), also known as the Public Docket Office, from noon to 4 p.m., Monday through Friday, excluding legal holidays. The TSCA NCIC is located at EPA Headquarters, Rm. NE–B607, 401 M St., SW., Washington, DC 20460.

List of Subjects in 40 CFR Part 372

Environmental protection, Chemicals, Community right-to-know, Reporting and recordkeeping requirements, and Toxic chemicals.

Dated: April 14, 1995.

Lynn R. Goldman,

Assistant Administrator for Prevention, Pesticides and Toxic Substances.

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GENERAL SERVICES ADMINISTRATION

48 CFR Parts 501, 503, 505, 506, 507, 552, and 570

[GSAR Notice 5–399]

RIN–AF67

General Services Administration Acquisition Regulation; Leasing Real Property

AGENCY: Office of Acquisition Policy, GSA.

ACTION: Proposed rule.

SUMMARY: The General Services Administration (GSA) invites written comments on a proposal to amend the General Services Administration Acquisition Regulation (GSAR) to implement various provisions of the Federal Acquisition Streamlining Act of 1994 as they apply to the acquisition of leasehold interests in real property and to implement recommendation of a GSA process re-engineering team for streamlining and/or improving the lease acquisition process.

DATES: Comments on the proposed rule should be submitted by June 19, 1995 to be considered in the formulation of the final rule.

ADDRESSES: Interested parties should submit written comments to Ms. Marjorie Ashby, General Services Administration, Office of GSA Acquisition Policy, 18th & F Streets, NW, Washington, DC 20405.

FOR FURTHER INFORMATION CONTACT: Tom Wiznowski, Office of GSA Acquisition Policy, (202) 501–1224.

SUPPLEMENTARY INFORMATION:

A. Background

This proposed rule implements several provisions of the Federal Acquisition Streamlining Act (FASA), Pub. L. 103–355, October 13, 1994 as it applies to the acquisition of leasehold interests in real property. Most of the provisions of FASA which are implemented in the Federal Acquisition Regulation (FAR) will also apply to leases of real property because the GSAR incorporates provision of the FAR that apply to leases of real property by reference. Other provisions of FASA are unique to leases of real property and are addressed in Part 570 of the GSAR. The most significant provisions of FASA that are implemented through changes in Part 570 are:

(1) Section 4402 of FASA amended the Federal Property and Administrative Services Act to authorize the Administrator of General Services to prescribe regulations that provide special simplified procedures for acquisitions of leasehold interests in real property at rental rates that do not exceed the simplified acquisition threshold. For purposes of establishing such procedures the rental rate or rates under a multiyear lease do not exceed the simplified acquisition threshold if the average annual rent payable for the period of the lease does not exceed the simplified acquisition threshold ($100,000).

(2) Section 1061 of FASA amended the Federal Property and Administrative Services Act to provide for disclosure of all significant evaluation factors and subfactors and to provide for disclosure to offerors whether all evaluation factors other than cost or price, when combined, are significantly more important than cost or price; approximately equal in importance to cost or price; or significantly less important than cost or price.

(3) Section 1063 of FASA amended the Federal Property and Administrative Services Act to provide for notification, in writing or by electronic means, of award to unsuccessful offerors within 3 days after the date of contract award.